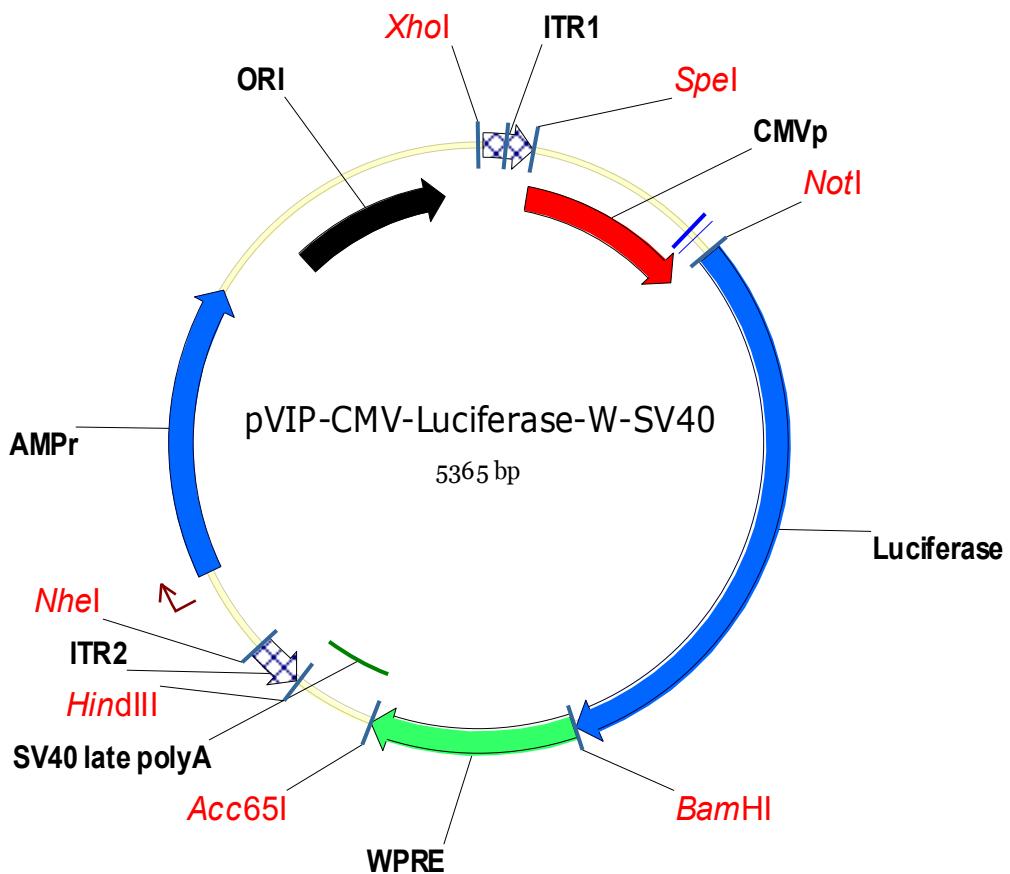


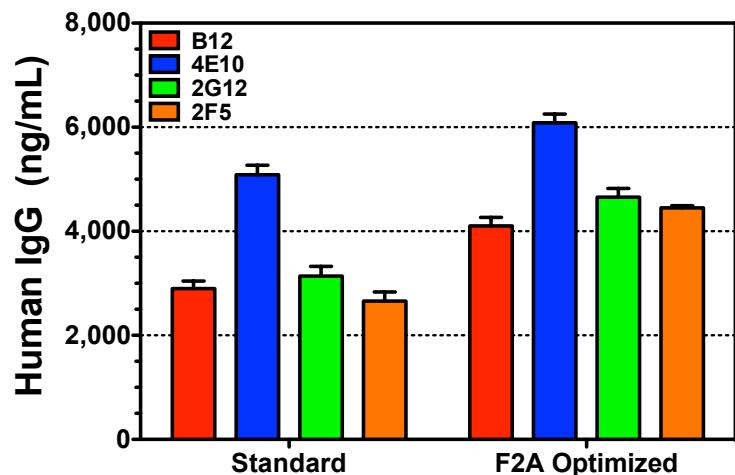
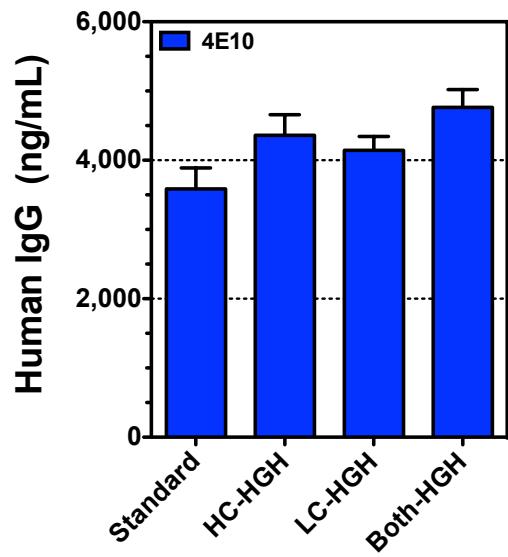
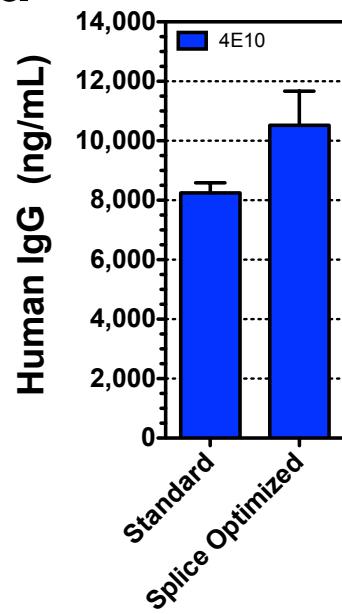
Supplementary Figure 1 – Development of a muscle-optimized AAV-based antibody expression vector

a, (left) Quantitation of luciferase activity by Xenogen imaging of Rag2^{-/-}/γc^{-/-} mice receiving intramuscular injection of 1x10¹⁰ or 1x10¹¹ GC of AAV2/8 encoding luciferase demonstrates long-term dose-dependent expression (n=2). (right) Concentration of human IgG in circulation as measured by total human IgG ELISA on serum samples taken after intramuscular injection of 1x10¹⁰ or 1x10¹¹ GC of AAV2/8 expressing 4E10-IgG1 into Rag2^{-/-}/γc^{-/-} mice (n=2). Antibody production is dose-dependent and is maintained for at least 64 weeks. **b**, Comparison of luciferase activity 15 weeks after intramuscular injection of 2x10⁹ GC of AAV2/8 vectors expressing luciferase from a panel of promoters (n=2). **c**, Design of the CASI promoter combining the CMV enhancer and chicken β-actin promoter followed by a splice donor (SD) and splice acceptor (SA) flanking the ubiquitin enhancer region. **d**, Comparison of luciferase activity from vectors driven by CASI as compared to conventional promoters 8 weeks after intramuscular injection of 1x10⁹ GC of AAV2/8 encoding luciferase driven by the indicated promoter (n=2). **e**, Comparison of luciferase activity 6 weeks post-administration of CMV-driven vectors with or without WPRE, terminated by the indicated polyadenylation signal (n=2). **f**, Schematic representation of the VIP expression vector for antibody expression indicating the inverted terminal repeats (ITR), the CASI promoter, an IgG1 heavy chain linked to kappa light chain separated by a self-processing 2A sequence, a WPRE for improved expression and SV40 late-polyadenylation signal. Antibody V-regions of heavy and light chains are cloned into the vector at positions indicated in red.



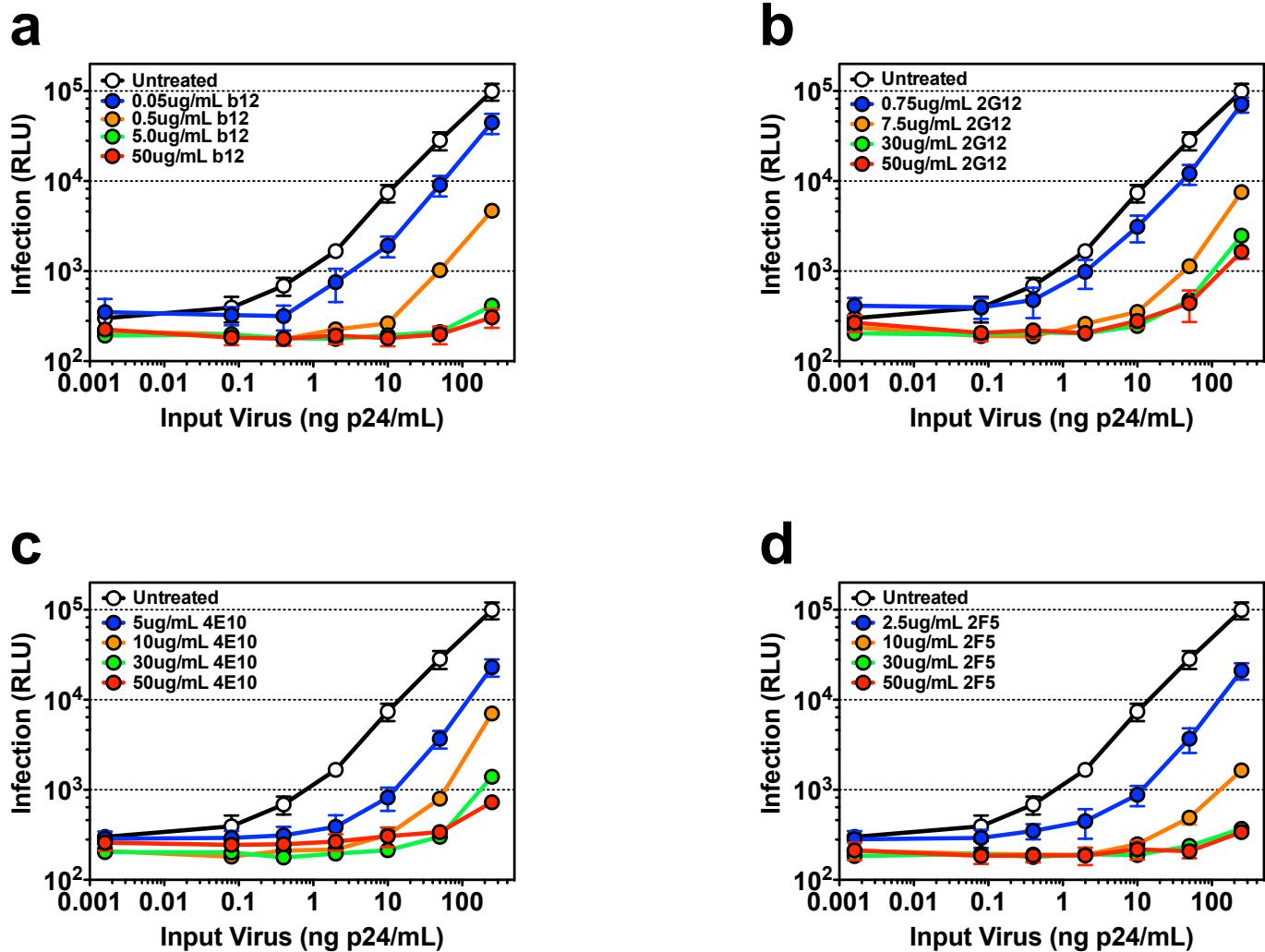
Supplementary Figure 2 – Map of the modular pVIP vector

Schematic representation of the pVIP transfer vector with unique restriction sites flanking each modular element designated in red. AAV sequences begin immediately following the *Xho*I restriction site with a 145bp “flip”-ITR from AAV2 followed by a *Spe*I restriction site and the immediate early CMV promoter. The promoter is followed by a *Not*I restriction site and one additional C residue to mimic a Kozak consensus sequence prior to the ATG of the luciferase transgene. The 3’ end of the transgene is terminated with a TAA stop codon followed by one additional A residue prior to the *Bam*HI site. The WPRE element follows this restriction site and continues until an *Acc65*I restriction site that precedes an SV40 polyadenylation signal and *Hind*III restriction site. Finally, a second 145bp AAV2 “flop”-ITR is located prior to an *Nhe*I site.

a**b****c****d**

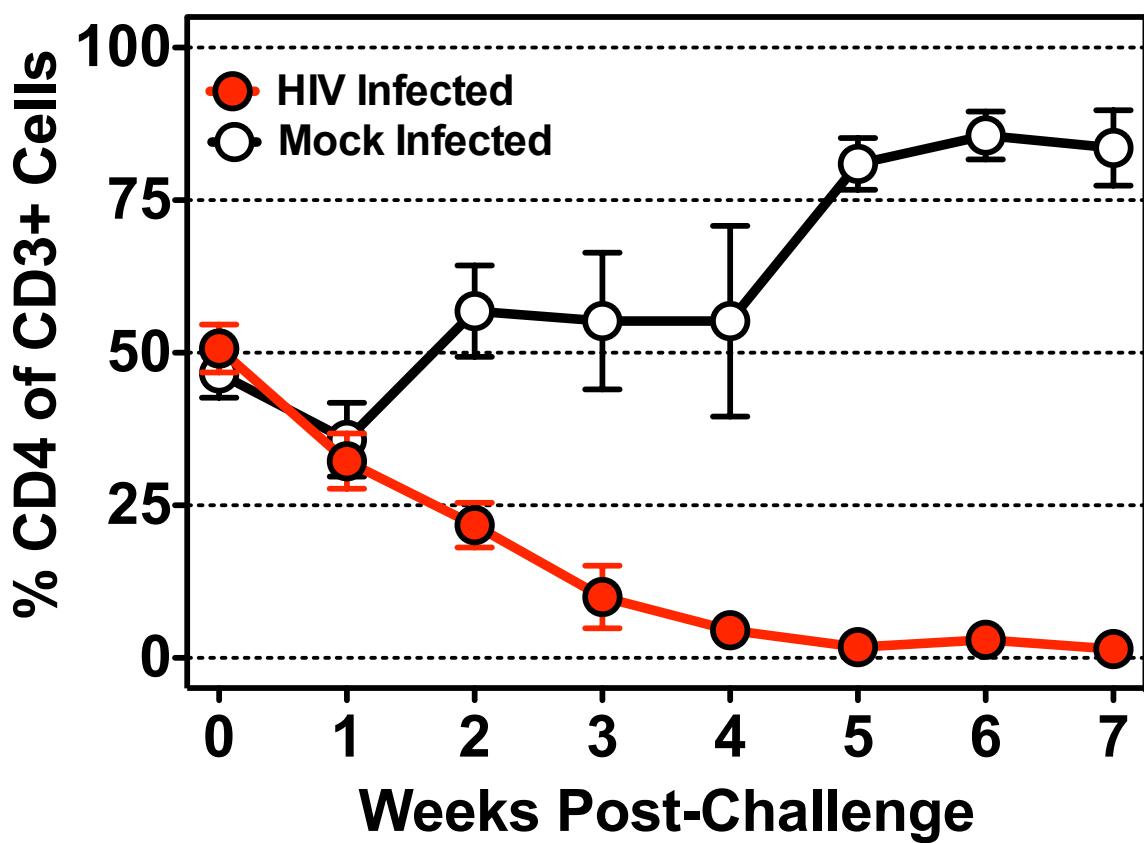
Supplementary Figure 3 – Optimization of the IgG1 transgene in vitro

a, Schematic representation of the IgG1 transgene that was optimized for expression in vitro. Highlighted are the heavy and light chain signal sequences (blue) the F2A self-processing peptide (green) and the predicted splice donor and acceptor sites (red lines). **b**, Comparison of antibody expression in vitro by ELISA following transfection with vectors carrying the antibody transgene shown above with standard or optimized F2A sequences that include a furin cleavage site. **c**, Comparison of 4E10 antibody expression in vitro by ELISA following transfection with vectors carrying 4E10 with natural or human growth hormone (HGH) derived signal peptides fused to the heavy chain gene, the light chain gene or both genes. **d**, Comparison of 4E10 antibody expression in vitro by ELISA following transfection with vectors carrying 4E10 in the standard expression cassette or a cassette in which the splice donors and acceptors were mutated to reduce the potential for extraneous splicing.



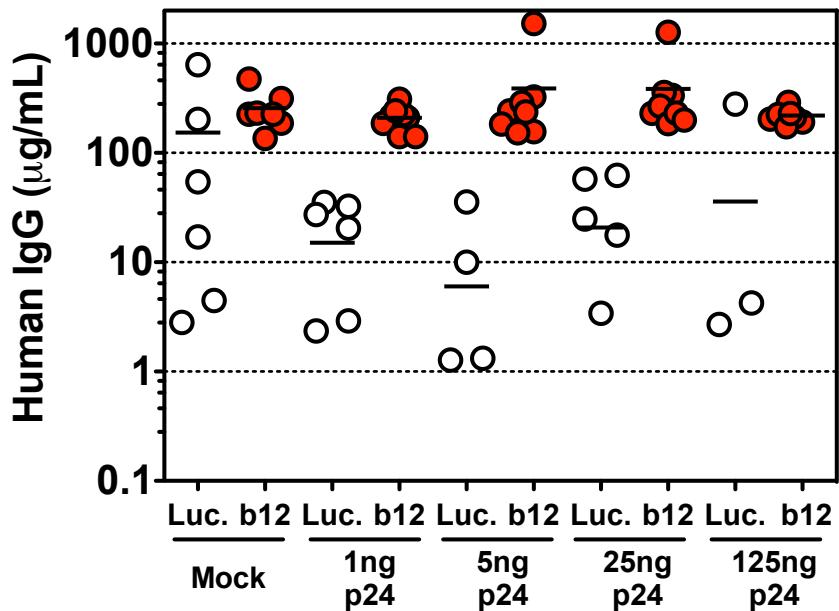
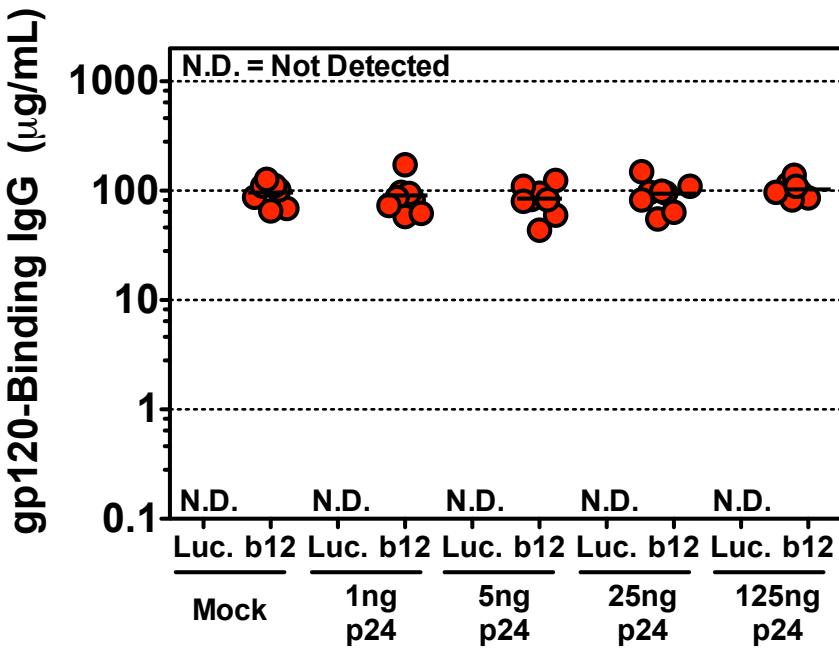
Supplementary Figure 4 – Neutralization of HIV by Antibodies expressed from an optimized expression transgene

To confirm that antibodies expressed from the optimized expression transgene retained their function, an in vitro protection assay using TZM-bl luciferase reporter cells was conducted. Cells were plated with the indicated concentrations of b12 (a), 2G12 (b), 4E10 (c) or 2F5 (d) prior to challenge with increasing titers of NL4-3. Two days after challenge, cells were lysed and quantitated for luciferase activity following the addition of luciferin substrate. (n=3, RLU=Relative luciferase Units).



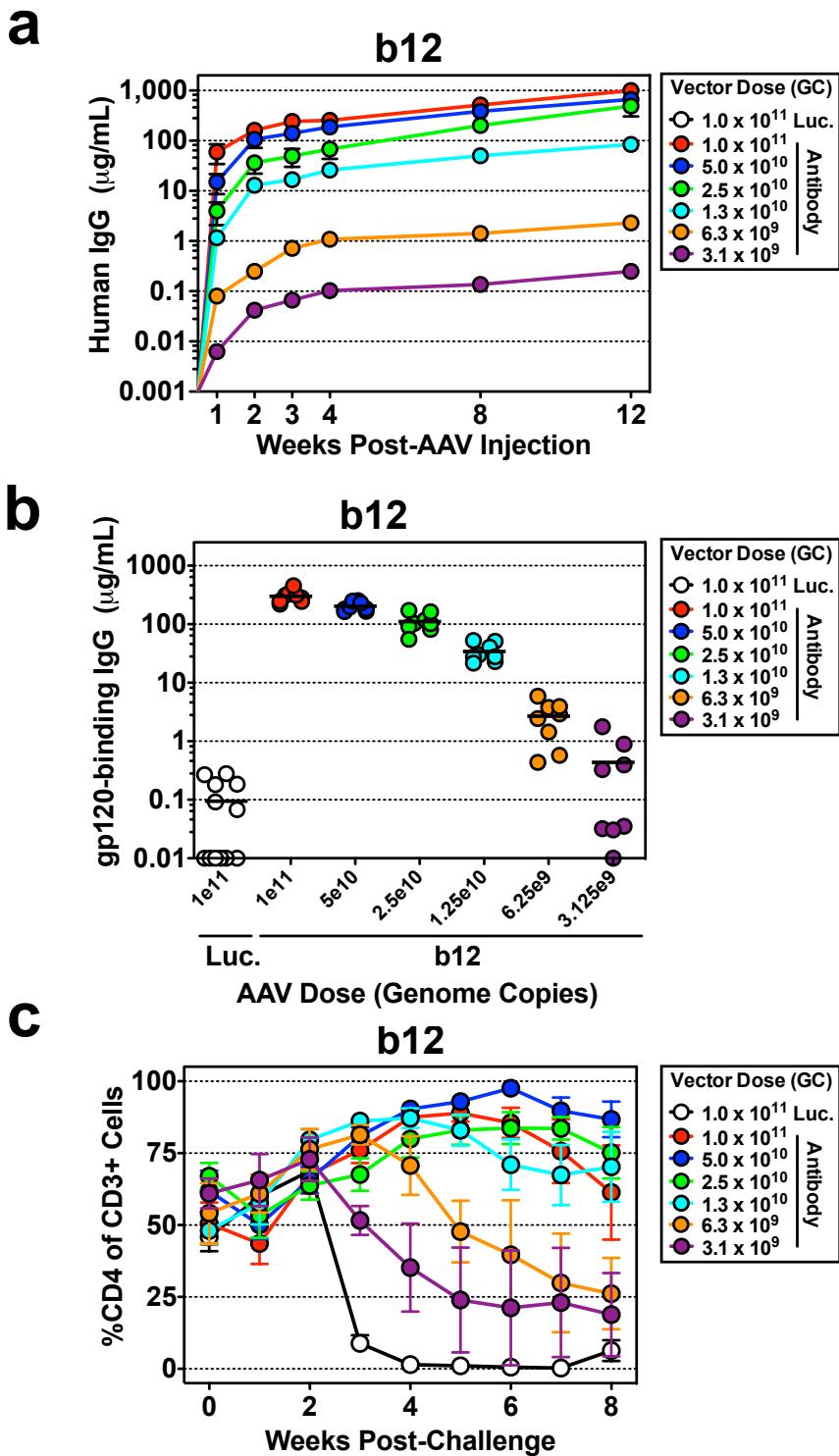
Supplementary Figure 5 – Depletion of CD4 cells in vivo following HIV challenge of humanized mice

Depletion of CD4+ T-cells in HuPBMC-NSG humanized mice following intraperitoneal (IP) challenge with 20ng p24 NL4-3 (n=4). To confirm that antibodies expressed from the optimized expression transgene retained their function, an in vitro protection assay using TZM-bl luciferase reporter cells was conducted. Cells were plated with the indicated concentrations of b12 (**a**), 2G12 (**b**), 4E10 (**c**) or 2F5 (**d**) prior to challenge with increasing titers of NL4-3. Two days after challenge, cells were lysed and quantitated for luciferase activity following the addition of luciferin substrate. (n=3, RLU=Relative luciferase Units).

a**b**

Supplementary Figure 6 – Serum concentrations of total human IgG and gp120 binding IgG prior to HIV challenge

a, Concentration of total human antibody produced by engrafted cells and VIP as measured by human IgG ELISA on serum samples taken 5 weeks after intramuscular injection of vectors expressing either luciferase or b12 antibody and 3 weeks after adoptive transfer of human PBMCs and the day prior to IV HIV challenge (n=8). **b**, Concentration of antibody at the same time point quantified using a gp120-specific ELISA to measure the concentration of antibody specific for HIV (n=8).



Supplementary Figure 7 – Determination of the minimum protective dose of b12 *in vivo*

a, b12 expression over time as a function of dose as determined by total human IgG ELISA on serum samples taken following AAV administration (n=8). Mice receiving luciferase-expressing vector exhibited no detectable human antibodies (n=12). **b**, Concentration of b12 in serum one day prior to challenge, 3 weeks after adoptive transfer of human PBMCs and 15 weeks after intramuscular administration of the indicated dose of AAV as determined by a gp120-specific ELISA to measure the fraction of antibodies capable of binding HIV (n=8-12). **c**, CD4 cell depletion in HuPBMC-NSG humanized mice as a result of intravenous challenge with 10ng of NL4-3 into animals expressing a range of b12 demonstrating the minimum dose of antibody necessary to protect against infection. Plots **a** and **c** show mean and standard error, plot **b** shows individual animals and mean (n=8-12).

Supplementary Materials

Optimal vectors for muscle-based antibody expression

To rapidly test novel vector configurations, we created a modular AAV transfer vector that implemented unique restriction sites flanking each modular element (Supplementary Fig. 2). To identify active promoters ideally suited to muscle expression, we created a series of vectors carrying the luciferase gene driven by a panel of ubiquitous and tissue-specific promoters. These vectors were administered intramuscularly via a single injection in the gastrocnemius muscle and luciferase expression was monitored to determine the relative expression potential of each promoter in this target tissue (Supplementary Fig. 1b). We identified the cytomegalovirus immediate early promoter (CMV), chimeric chicken- β -actin (CAG), and ubiquitin C (UBC) promoters as optimal for long-term muscle expression. Based on these findings, we created a novel promoter that combined these three promoters along with consensus splice donor and acceptor sequences to produce the CASI promoter design (Supplementary Fig. 1c). Further *in vivo* testing demonstrated that the CASI promoter was considerably more active in muscle than the CAG promoter despite being 34% more compact (Supplementary Fig. 1d). This reduced size allowed us to incorporate the woodchuck hepatitis virus posttranscriptional regulatory element (WPRE)³⁰, which we confirmed to significantly enhance expression of transgenes (Supplementary Fig. 1e). To determine the relative efficiency of polyadenylation signals for muscle-derived expression, we tested the SV40 late poly(A), the rabbit beta-globin (RBG) poly(A) and the bovine growth hormone (BGH) poly(A), all of which demonstrated comparable levels of expression (Supplementary Fig. 1e). Based on these results, we designed a muscle-optimized expression vector encoding an IgG1

scaffold into which heavy and light chain V-regions derived from monoclonal antibodies could be inserted (Supplementary Fig. 1f).

Optimization of the antibody transgene

To create an optimal framework for the expression of antibody, we cloned the heavy and light chains of several broadly neutralizing HIV antibodies separated by an F2A self-processing peptide sequence³¹ (Supplementary Fig. 3a) into a mammalian expression vector under the control of the CMV promoter. 293T cells transfected with these vectors demonstrated secretion of human IgG into the culture supernatant that could be detected by ELISA (Supplementary Fig. 3b). In an attempt to improve expression, we re-engineered the F2A sequence to better reflect mammalian codon usage and incorporated a furin cleavage site at the N-terminus for optimal processing²². Comparison of these F2A optimized vectors by transfection showed they produced higher levels of all four antibodies tested.

We next sought to improve secretion of antibody by replacing the endogenous signal sequences with a codon optimized sequence derived from the well-characterized human growth hormone (HGH) and created versions of the 4E10 expression vector in which either the heavy chain, the light chain, or both chains were driven by separate HGH signal sequences and compared their expression by transfection. To minimize repetitive sequence in our viral vectors, two HGH sequences were synthesized which had distinct nucleotide sequences but encoded identical amino acids, and each were used for either the heavy or light chain exclusively. Replacement of the endogenous signal sequences with HGH sequences at either the heavy or light chains resulted in higher

levels of antibody production, and signal sequence replacement of both chains yielded the best results (Supplementary Fig. 3c).

To remove the potential for inappropriate splicing of the transcript encoding the antibody, we subjected the sequence to *in silico* splice prediction³² and removed all potential splice donor and acceptor sequences through the use of conservative mutations to the site or, when this was not possible, the surrounding sequences. We observed improved expression of the 4E10 antibody when placed in this splice-optimized framework (Supplementary Fig. 3d). The final antibody transgene consists of an HGH signal sequence followed by a swappable V_H region, a splice-optimized heavy chain constant region, a furin cleavage site linked to an optimized F2A peptide which is fused to a second HGH signal sequence, a swappable V_L region, and a splice-optimized kappa light chain constant region.

Validation of transgene-expressed antibody activity

To confirm that the optimizations made to improve gene expression did not impact the neutralizing efficacy of the antibodies, we expressed several well-studied broadly neutralizing antibodies from this expression cassette and tested these purified proteins in an *in vitro* protection assay. Cells carrying a luciferase gene under the control of HIV-induced transcriptional elements (TZM-bl cells) were incubated with dilutions of each antibody prior to challenge with increasing amounts of HIV. We observed robust reduction in TZM-bl cell infection at antibody concentrations that correlated well with the previously established IC₅₀ and IC₉₀ values for all antibodies tested against this strain (Supplementary Fig. 4).

Methods

Construction and cloning of modular AAV transfer vectors

To construct the AAV transfer vector, oligonucleotides encoding the 145bp AAV2-derived ITR1 in the flip orientation and ITR2 in the flop orientation flanked by unique restriction sites were synthesized (Integrated DNA Technologies) and annealed prior to ligation into PBR322 plasmid vector. Subsequently, promoters, transgenes and polyadenylation signals flanked by compatible sites were amplified by PCR and cloned between the ITRs, resulting in a modular AAV transfer vector in which unique combinations of restriction sites flanked each element.

AAV virus production and purification

AAV8 was purified from culture supernatants as described^{33,34} with some modifications. 293T cells were maintained in DMEM medium supplemented with 10% fetal bovine serum, 1% penicillin-streptomycin mix (Mediatech), and 1% glutamine (Mediatech) in a 5% CO₂ incubator at 37°C. Three days prior to transfection, four 15cm plates were seeded with 3.75×10^6 cells each in 25mL media. Two hours prior to transfection, media was changed to 15mL of fresh media. The AAV backbone vector was co-transfected with helper vectors pHELP (Applied Viromics) and pAAV 2/8 SEED (University of Pennsylvania Vector Core) at a 0.25:1:2 ratio using BioT transfection reagent (Bioland Scientific). The total amount of DNA used per transfection was 80µg. Five AAV virus collections were performed at 36, 48, 72, 96, and 120 hours post-transfection. For each time point, media was filtered through a 0.2µm filter and 15mL of fresh media was gently added to the plate. After collection, approximately 75mL of 5X PEG solution (40% polyethylene glycol, 2.5M NaCl) was added to the total volume of supernatant collected (~300mL) and the virus was precipitated on ice for at least two hours³⁵. Precipitated virus was pelleted at 5,000 rpm for 30min (Sorvall RC 3B Plus) and resuspended in 1.37g/mL cesium chloride. Resuspended virus was split evenly into two Quick-Seal tubes (Beckman) and spun at 60,000 rpm at 20°C for 24 hours (Beckman Coulter, Optima LE-80K, 70Ti rotor). Fractions of 100–200µL were collected in a 96-well flat-bottom tissue culture plate, and a refractometer was used to quantitate the refractive index of 5µL of each fraction. Wells exhibiting refractive indexes between 1.3755-1.3655 were combined and diluted to a final volume of 15mL using Test Formulation Buffer 2 (TFB2, 100mM sodium citrate, 10mM Tris, pH8)³⁶. Virus was loaded onto 100kDa MWCO centrifugal filters (Millipore) and subjected to centrifugation at 1,400 rpm at 4°C until 1mL retentate remained. Retained virus was then again diluted to 15mL total volume in TFB2 and this process was repeated such that the virus was washed three times. Final retentate volume was between 500-1000µL total, which was aliquoted and stored at -80°C.

AAV quantification and functional validation

Purified AAV was quantified by qPCR essentially as described³⁷ with the following modifications. Frozen aliquots of AAV were thawed and diluted ten-fold in digestion buffer containing 10 units of DNase I (Roche) and incubated at 37°C for 30 minutes. DNase digested virus was serially diluted and 5µL of each dilution was used in a 15µL

qPCR reaction with PerfeCTa SYBR Green SuperMix, ROX (Quanta Biosciences) and primers designed against the CMV enhancer (5' CMV: AACGCCAATAGGGACTTCC and 3' CMV: GGGCGTACCTGGCATATGAT) or the luciferase transgene (5' Luc: ACGTGCAAAAGAACGCTACCG and 3' Luc: AATGGGAAGTCACGAAGGTG). Samples were run in duplicate on an Applied Biosystems 7300 Real Time PCR System. The following cycling conditions were used: 1 cycle of 50°C for 2min, 1 cycle of 95°C for 10min, 40 cycles of 95°C for 15s and 60°C for 60s. Virus titer was determined by comparison with a standard curve generated using either a purified DNA fragment cut with XhoI/NheI from the pVIP luciferase expressing vector or a reference standard consisting of purified AAV2/8 expressing 4E10 antibody previously titrated against the DNA standard.

To validate the functional activity of each lot of the titered virus we carried out *in vitro* infection assays using 293T cells and measured the concentration of the antibody in the cell supernatant. Twenty-four hours prior to infection, 12-well plates were seeded with 500K cells in 1mL of media. Two hours before infection, media was replaced with 500 μ L per well of fresh media. 10¹¹ genome copies of each virus were added to each well and allowed to infect for six days. Supernatants were removed and quantitated for total IgG production by ELISA.

Mouse strains

Immunodeficient NOD/SCID/ γ c^{-/-} (NSG), immunocompetent c57BL/6 (B6), and Balb/C mice were obtained from the Jackson Laboratory (JAX). Immunodeficient Rag2^{-/-} γ c^{-/-} mice were obtained from Dr. A. Berns at the Netherlands Cancer Institute.

AAV intramuscular infection and bioluminescent imaging

Aliquots of previously titered viruses were thawed slowly on ice and diluted in TFB2 to achieve the predetermined dose in a 40 μ L volume. Mice were anesthetized by isofluorane inhalation and a single 40 μ L injection was administered into the gastrocnemius muscle with a 28G insulin syringe. At various times following vector administration, mice were either bled to determine antibody concentration in serum or imaged using a Xenogen IVIS 200 Series imaging system (Caliper Lifesciences). To image, mice were anesthetized by isofluorane inhalation and given 100 μ L of 15mg/mL D-luciferin (Gold Biotechnology) via intraperitoneal injection. Images were taken between 5 and 10 minutes after D-luciferin injection.

Quantitation of antibody production by ELISA

For detection of total human IgG, ELISA plates were coated with 1 μ g per well of goat anti-human IgG-Fc antibody (Bethyl) for 1 hour. Plates were blocked with 1% BSA (KPL) in TBS for at least 2 hours. Samples were incubated for 1 hour at room temperature in TBST containing 1% BSA (KPL), then incubated for 30 min with HRP-conjugated goat anti-human kappa light chain antibody (Bethyl). Sample was detected with TMB Microwell Peroxidase Substrate System (KPL). A standard curve was generated using either Human Reference Serum (Lot 3, Bethyl) or purified Human IgG/Kappa (Bethyl).

For detection of gp120-binding IgG, ELISA plates were coated with 0.04-0.10 µg per well HIV-1 gp120MN protein (Protein Sciences) for 1 hour. Plates were blocked with 1% BSA (KPL) in TBS for at least 2 hours. Samples were incubated for 1 hour at room temperature in TBST containing 1% BSA (KPL), then incubated for 30 min with HRP-conjugated goat anti-human IgG-Fc antibody (Bethyl). Sample was detected with TMB Microwell Peroxidase Substrate System (KPL). A standard curve was generated using either purified b12 or VRC01 protein as appropriate for the samples.

HIV virus production and titering

293T cells were maintained in DMEM medium supplemented with 10% fetal bovine serum, 1% penicillin-streptomycin mix(Mediatech), 1% glutamine (Mediatech) in a 5% CO₂ incubator at 37°C. Three days prior to transfection, two 15cm plates were seeded with 3.75×10^6 cells each in 25mL media. Two hours prior to transfection, media was changed to 15mL of new media. 40µg of the pNL4-3 plasmid³⁸ encoding an infectious molecular clone of HIV was transfected using Trans-IT reagent (Mirus) according to manufacturers' instructions. Supernatant collections were performed at 24, 48, and 72 hours post transfection and 15mL of fresh media was gently added back to plate after each harvest. Pooled supernatants were filtered using a 0.45µm filter to remove cell debris and aliquoted for storage at -80°C. HIV was quantified following the manufacturers' instructions using the Alliance HIV-1 p24 antigen ELISA kit (Perkin-Elmer).

***In vitro* HIV protection assay**

In vitro neutralization assays in luciferase reporter cells were performed as described³⁹ with the following modifications. TZM-bl cells from the NIH AIDS Research and Reference Reagent Program were maintained in DMEM medium supplemented with 10% fetal bovine serum, 1% penicillin-streptomycin mix (Mediatech), 1% glutamine (Mediatech) in a 5% CO₂ incubator at 37°C. Prior to the assays, TZM-bl cells were trypsinized, counted, and resuspended in a concentration of 10^5 cells/mL, in a total volume of 15mL. Cells were mixed with 75µg/mL DEAE-Dextran and varying concentrations of each antibody as indicated and allowed to incubate on ice during the preparation of the virus. To prepare virus dilutions, stock NL4-3 was diluted to 250ng/mL in growth media and subsequently four-fold serially diluted in the assay plate. 100µL of media containing 10,000 cells pre-incubated with antibody were added to wells containing previously diluted virus. Infection was allowed to proceed for 48 hours in a 5% CO₂ incubator at 37°C. Prior to reading the plate, 100µL of BriteLite reagent (Perkin Elmer) was added to each well, and the plate was incubated for two minutes at room temperature. 120µL of each well was then transferred to an opaque plate and read by the VICTOR3 (Wallac 1420 VICTOR3 plate reader, PerkinElmer).

Production of humanized mice for *in vivo* challenge

Humanized mice were produced essentially as described¹⁷ with the following modifications. Human peripheral mononuclear blood cells (AllCells) were thawed from -80°C, expanded in RPMI medium supplemented with 10% fetal bovine serum, 1% penicillin-streptomycin mix(Mediatech),1% glutamine (Mediatech), 50µM beta-

mercaptoethanol, 10mM HEPES(Gibco), 1X non-essential amino acids (Gibco), 1X sodium pyruvate (Gibco), and stimulated for T cell expansion with 5 μ g/mL phytohemagglutinin (Sigma) and 10ng/mL human IL-2 (Peprotech) in a 5% CO₂ incubator at 37°C. Cells were expanded for 7-13 days prior to use. For engraftment, 2-4 million cells were injected IP into NSG mice in a 300 μ L volume of media.

HIV protection experiments

One day prior to HIV challenge, blood samples from each mouse were subjected to both ELISA for antibody quantitation as well as flow cytometry to determine baseline CD4/CD8 ratios. The following day, mice were challenged through either IP or IV injection of 100 μ L containing the specified dose of HIV diluted in PBS. Infected mice were subjected to weekly blood sampling to determine the ratio of CD4 to CD8 cells in the T lymphocyte subset by flow cytometry.

Flow cytometry

Blood samples were taken from mice by retro-orbital bleeding and were centrifuged for 5 minutes at 3,500 rpm in a microcentrifuge to separate plasma from cell pellets. Plasma was removed and frozen for future analysis and cell pellets were resuspended in 1.1mL of 1X RBC lysis buffer (Biolegend) and incubated on ice for at least 10 minutes to remove red blood cells. After lysis, samples were pelleted at 3,500 rpm in a microcentrifuge for 5 minutes at room temperature, and stained with 65 μ L of a cocktail containing 5 μ L anti-human CD3-FITC, 5 μ L anti-human CD4-PE, and 5 μ L anti-human CD8a-APC antibodies (Biolegend) and 50 μ L of phosphate buffered saline supplemented with 2% fetal bovine serum (PBS+). Samples were washed with 1mL PBS+ and again pelleted at 3,500 rpm in a microcentrifuge for 5 minutes. Pelleted cells were resuspended in 200 μ L of PBS+ supplemented with 2 μ g/mL propidium iodide (Invitrogen), and analyzed on a FACSCalibur flow cytometer (Beckton-Dickenson). Samples were first gated by CD3 expression prior to determining the ratio of CD4 to CD8 cells within this subset. Samples containing fewer than 20 CD3+ events were excluded from the analysis.

Histological staining for HIV p24

At the conclusion of the *in vivo* challenge experiments, spleens were removed from mice and immersed in 10% neutral buffered formalin for 24 hours. Following fixation, tissues were removed and placed in 70% ethanol until standard paraffin embedding and processing. Four-micron thick sections were then taken and immunohistochemical (IHC) staining was performed for HIV-p24 detection using the Kal-1 murine monoclonal antibody and standard antigen retrieval techniques⁴⁰. The slides were reviewed by a pathologist (D.S.R.) on an Olympus BX51 light microscope and images obtained using a SPOT Insight Digital Camera (Diagnostic Instruments).

Supplementary References

- 30 Zufferey, R., Donello, J. E., Trono, D. & Hope, T. J. Woodchuck hepatitis virus posttranscriptional regulatory element enhances expression of transgenes delivered by retroviral vectors. *J Virol* **73**, 2886-2892 (1999).
- 31 Szymczak, A. L. *et al.* Correction of multi-gene deficiency in vivo using a single 'self-cleaving' 2A peptide-based retroviral vector. *Nat Biotechnol* **22**, 589-594, doi:10.1038/nbt957 (2004).
- 32 Reese, M. G., Eeckman, F. H., Kulp, D. & Haussler, D. Improved splice site detection in Genie. *Journal of computational biology : a journal of computational molecular cell biology* **4**, 311-323 (1997).
- 33 Lock, M. *et al.* Rapid, simple, and versatile manufacturing of recombinant adeno-associated viral vectors at scale. *Human gene therapy* **21**, 1259-1271, doi:10.1089/hum.2010.055 (2010).
- 34 Ayuso, E. *et al.* High AAV vector purity results in serotype- and tissue-independent enhancement of transduction efficiency. *Gene therapy* **17**, 503-510, doi:10.1038/gt.2009.157 (2010).
- 35 Matsushita, T. *et al.* Adeno-associated virus vectors can be efficiently produced without helper virus. *Gene therapy* **5**, 938-945, doi:10.1038/sj.gt.3300680 (1998).
- 36 Wright, J. F. *et al.* Identification of factors that contribute to recombinant AAV2 particle aggregation and methods to prevent its occurrence during vector purification and formulation. *Mol Ther* **12**, 171-178, doi:10.1016/j.ymthe.2005.02.021 (2005).
- 37 Rohr, U. P. *et al.* Fast and reliable titration of recombinant adeno-associated virus type-2 using quantitative real-time PCR. *Journal of virological methods* **106**, 81-88 (2002).
- 38 Adachi, A. *et al.* Production of acquired immunodeficiency syndrome-associated retrovirus in human and nonhuman cells transfected with an infectious molecular clone. *J Virol* **59**, 284-291 (1986).
- 39 Montefiori, D. C. Evaluating neutralizing antibodies against HIV, SIV, and SHIV in luciferase reporter gene assays. *Current protocols in immunology / edited by John E. Coligan ... [et al.] Chapter 12*, Unit 12 11, doi:10.1002/0471142735.im1211s64 (2005).
- 40 Kaluza, G. *et al.* A monoclonal antibody that recognizes a formalin-resistant epitope on the p 24 core protein of HIV-1. *Pathology, research and practice* **188**, 91-96 (1992).